Role of Multimodality Intraoperative Neurophysiological Monitoring during Embolisation of a Spinal Cord Arteriovenous Malformation

A Paradigmatic Case

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Key words: Motor evoked potentials, somatosensory evoked potentials, neurophysiological monitoring, embolisation, spinal arteriovenous malformations

Summary

The decision whether or not to embolise during endovascular procedures for arteriovenous malformations (AVMs) of the spinal cord under general anesthesia, relies primarily on neurophysiological results of provocative tests with Lidocaine and short-acting barbiturates. Because of the complex haemodynamics of spinal AVMs, when either sensory (CSEPs) or muscle motor evoked potentials (mMEPs) are used independently, they can mislead the interpretation of provocative tests. This report illustrates the specific but complementary role played by provocative tests using CSEPs and mMEPs during embolisation of a low thoracic spinal cord AVM.

We present the case of a 46 year old male with six year history of right lower extremity weakness. At that time, Magnetic Resonance (MR) imaging of the spine disclosed an intramedullary AVM at T11. He remained neurologically stable up to seven months before admission, when he developed sudden onset of low back pain, followed by progressive paraparesis, numbness in lower extremities, urinary retention and fecal incontinence. A new MR imaging study indicated venous thrombosis of the AVM.

A two-stage embolisation was performed. During the first procedure, after provocative tests did not affect either CSEPs or mMEPs, an embolisation was performed through a sulcocommisure feeder from the anterior spinal artery (ASA) at T9. Conversely, provocative tests with Lidocaine performed from a right posterior spinal artery (PSA) feeder to the AVM nidus resulted in a significant (>50%) decrease of CSEPs, while mMEPs remained unchanged. The repeatedly positive tests warranted further investigation of the vascular anatomy which disclosed a normal right PSA distal to the nidus; the distal normal PSA was protected with coils. A repeated Lidocaine test was negative and the posterior feeder was embolised with no subsequent changes in CSEPs or mMEPs. After the procedure, the patient experienced only a mild transitory increase in right leg numbness, but no additional motor deficits. Five days later, the embolisation through the ASA feeder at T9 was completed on the basis of negative provocative tests. No additional neurological deficits were observed.

Favoring either CSEPs or MEPs during endovascular procedures in the spinal cord is not justified by a solid scientific background. This case report illustrates that monitoring both CSEPs and mMEPs combined with provocative tests allows the safest and most effective embolisation of spinal cord AVMs under general anesthesia.

Introduction

In order to avoid ischemic complications during endovascular treatment of spine and spinal vascular lesions, a clear understanding of the angioarchitecture of the lesion and the normal surrounding structures is critical. The ability to assess neurological function with the use of provocative testing and detailed examination during the procedure can provide additional safety, though it can never supplant a careful anatomically-based angiographic study. Intraoperative neurophysiological monitoring has been used over the last 15 years to assess the functional integrity of the spinal cord during endovascular procedures under general anesthesia.

Since the early eighties, cortical somatosensory evoked potentials (CSEP) have been used by our and other groups [1,5,6] to directly monitor the sensory pathways as well as to obtain indirect information on the functional integrity of the corticospinal tracts. In the senior author's (AB) experience, the introduction of CSEP monitoring and the use of provocative tests (e.g. intra-arterial administration of Lidocaine and short acting barbiturates) played a significant role in dramatically reducing the incidence of complications from selective spinal angiography and embolisation from 20% to less than 2% [1,6].

The anterior spinal artery (ASA) accounts for four-fifths of spinal cord vascular supply, including the substantia gelatinosa and a portion of dorsal columns [7]. This observation led to the assumption that an ischemic injury affecting descending motor pathways would have produced concurrent changes in the dorsal column activity, ultimately resulting in changes of CSEPs. Berenstein et Al [1] consistently observed significant decreases in CSEP amplitude after transient vascular occlusion of the ASA. In some cases, CSEP changes even anticipated the appearance of motor deficits.

Unfortunately, the early expectation for the reliability and sensitivity of CSEPs as rapid indicators of compromised spinal cord blood flow has been partially disappointed. Deterioration in motor function despite unchanged intraoperative CSEPs, after spine and spinal cord surgery [6,8,9] as well as following embolisation of spinal cord arteriovenous malformations (AVM) [10], has been reported. While reports on the use of motor evoked potentials (mMEPs) during endovascular procedures remain anecdotal [11,12], we recently reported on correlations of mMEPs with angiographic findings of impaired ASA blood flow [13], and on mMEPs' prognostic value during embolisation of a spinal dural arteriovenous fistula [14].

However, when either CSEPs or mMEPs are used independently, they can mislead the interpretation of provocative tests and, consequently, the decision-making process whether on or not to proceed with embolisation. This report illustrates the specific but complementary role played by CSEP and mMEPs during embolisation of a low thoracic spinal cord AVM. The role of neurophysiological monitoring and provocative tests under general anesthesia is discussed.

Case Report

Clinical History

This is a 46-year-old male with an unremarkable medical history up to six years before admission to our Institution when he started complaining of right lower extremity weakness. At that time, a spine MRI disclosed intramedullary signal abnormalities at T11 indicative of an intramedullary AVM. He received no treatment and remained neurologically stable for almost five years. Seven months prior to admission, he developed sudden onset of low back pain, radiating down to the inner aspect of the left leg and distally to the left toes. Pain was followed by progressive paraparesis, urinary retention and fecal incontinence.

Magnetic resonance (MR) imaging demonstrated expansion of the cord at T11-12 and signal abnormalities highly suspicious for venous thrombosis of the intramedullary AVM. Over the following months, the patient's symptoms gradually improved. The patient was transferred to our Institution for endovascular treatment.

Positive neurological findings on admission were: spasticity and motor weakness on both lower extremities, foremost distally and on the right side (tibialis anterior 0-1/5; extensor hallucis longus 3/5). Decreased pin sensation from L5 to S5 on the left, and decreased vibration sense on both feet, left more than right. The patient had control of the anal sphincter but still had urinary retention.

Material, Methods and Results

In our Institution, all spinal endovascular procedures are routinely performed under general anesthesia. In order to perform proper neurophysiological monitoring, continuous infusion of propofol (100-150 μg/kg/min) and fentanyl (1 μg/kg/h) with no halogenated agents is used throughout the procedure. No muscle relaxants are given except short acting relaxants for intubation.

Neurophysiological Monitoring

In consideration of both the location of the AVM and the clinical evidence of bowel/bladder dysfunction, the bulbocavernosus reflex was also monitored in addition to CSEPs and mMEPs from upper and lower extremities.

CSEPs were elicited by stimulation of the posterior tibial nerve at the ankle (intensity 40 mA, duration 0.2 ms, repetition rate of 4.3 Hz). Recordings were performed via corkscrew-like subcutaneously inserted electrodes in the scalp (CS electrode, Neuromedical Inc., Herndon, VA) at CZ'- FZ according to the 10-20 International EEG system. As a control modality, we elicited CSEPs from both median nerves by stimulation at wrist (intensity 20 mA, duration 0.2ms, repetition rate of 4.3 Hz), recording via corkscrew-like electrodes from the scalp at C3'/C4'-CZ'.

Muscle MEPs were elicited with transcranial electrical stimulation of the motor cortex using corkscrew-like elec-

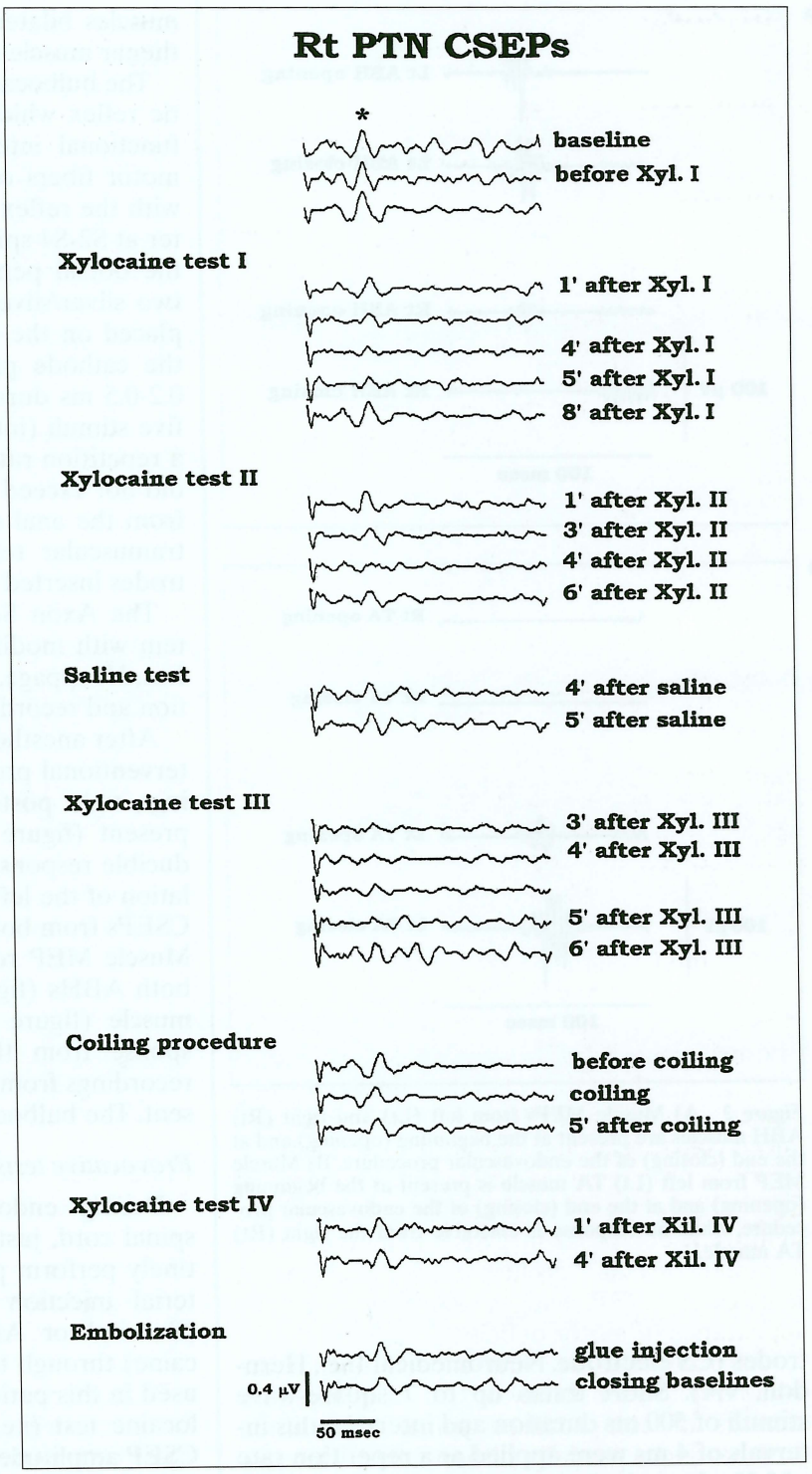

Figure 1 The right posterior tibial nerve CSEP monitored throughout the procedure. The amplitude (*) consistently decreased more than 50% in three consecutive provocative tests with Xylocaine (Xylocaine tests I-III), while it remained unchanged after saline injection. During the coiling procedure, and the following provocative test (Xylocaine test IV), CSEP amplitude remained stable. No changes in CSEPs were observed during embolisation of the AVM (glue injection) and closing baselines resembled those recorded at the beginning of the procedure.

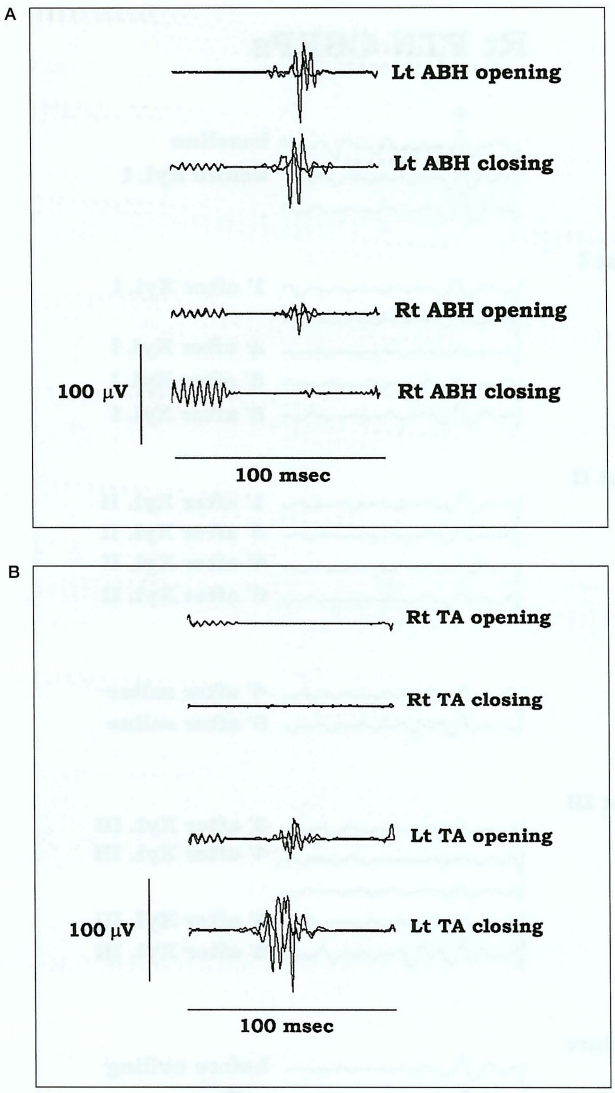

Figure 2 A) Muscle MEPs from left (Lt) and right (Rt) ABH muscles are present at the beginning (opening) and at the end (closing) of the endovascular procedure. B) Muscle MEP from left (Lt) TA muscle is present at the beginning (opening) and at the end (closing) of the endovascular procedure, while no response is elicitable from the right (Rt) TA muscle.

trodes (CS electrode, Neuromedical Inc., Herndon, VA). Short trains up to 7 square-wave stimuli of 500 ms duration and interstimulus intervals of 4 ms were applied at a repetition rate of 2 Hz through electrodes placed at C1 and C2 scalp sites, according to the International 10/20 EEG System. The stimulation intensity did not exceed 160 mA. Muscle responses were recorded via needle electrodes inserted into the anterior tibial (TA) and abductor hallucis (ABH) muscles bilaterally. Recordings from the right thenar muscle served as control[15].

The bulbocavernosus reflex is an oligosynaptic reflex which allows the operator to assess functional integrity of both the sensory and motor fibers of the pudendal nerves together with the reflex center located in the gray matter at S2-S4 spinal segments. For stimulation of the dorsal penile nerve (pudendal afferents), two silver/silver chloride disc electrodes were placed on the dorsal aspect of the penis with the cathode proximal. Rectangular pulses of 0.2-0.5 ms duration were applied as a train of five stimuli (interstimulus intervals of 4 ms) at a repetition rate of 2.3 Hz. Stimulus intensities did not exceed 40 mA. Recordings were made from the anal sphincter using two pairs of intramuscular teflon-coated hooked wire electrodes inserted in anal hemisphincters[16].

The Axon Sentinel-4 evoked potential system with modified software (AXON Systems, Inc., Hauppage, NY) was used for both stimulation and recording.

After anesthesia induction but before any interventional procedure, at the baseline recordings, right posterior tibial nerve CSEPs were present (figure 1, baseline), while no reproducible responses were obtainable after stimulation of the left posterior tibial nerve. Control CSEPs from both median nerves were present. Muscle MEP responses were obtainable from both ABHs (figure 2A) and from the left TA muscle (figure 2B), while there was no response from the right TA muscle. Control recordings from right thenar muscles were present. The bulbocavernosus reflex was present.

Provocative tests

During endovascular procedures for the spinal cord, just before embolisation, we routinely perform provocative tests with intra-arterial injection of short acting barbiturates (Brevitol or Amytal) and Lidocaine (Xylocaine) through the microcatheter. Brevitol was used in this patient. A positive Brevitol or Xylocaine test (i.e. more than 50% decrease in CSEP amplitude and/or mMEP disappearance) indicates that the vessel distal to the tip of the microcatheter supplies functional gray or white matter of the spinal cord respectively. We do not perform embolisation with liquid embolic material from the same catheter position where provocative tests was positive.

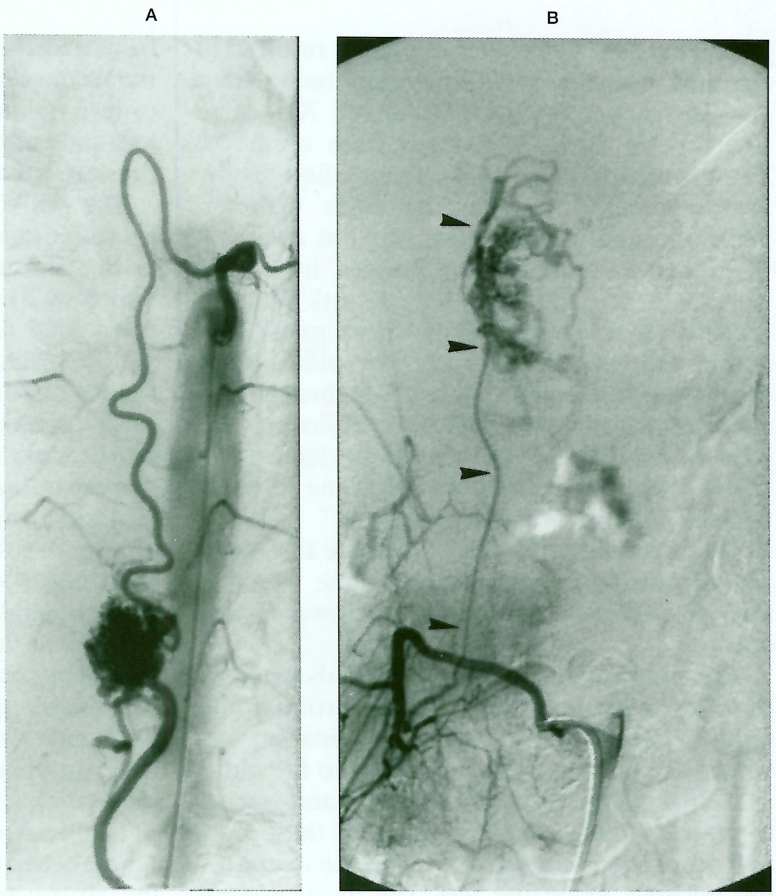

Figure 3 A) Left T9 intercostal artery angiogram, demonstrating a hypertrophied anterior spinal artery supplying an intramedullary nidus type arteriovenous malformation at the T12 level with a dilated vein draining the malformation caudally. B) Right L2 lumbar artery angiogram demonstrating hypertrophied posterior spinal artery (PSA) (arrow heads) supplying the lateral portion of the nidus of the AVM. The same portion of the nidus is supplied by the PSA from the right T11 intercostal artery (not shown).

Endovascular procedures

Angiographic study

The angiographic study demonstrated a markedly hypertrophied radiculomedullary artery originating from the left T9 intercostal artery and supplying the AVM at the T12 level. At least three nidal aneurysms and two venous aneurysms were seen (figure 3A). There was also evidence of spinal cord venous hypertension in the lower spinal cord and conus. The venous drainage of the malformation consisted of a markedly dilated anterior spinal vein caudally, and a slow-flow drainage on the posterior surface of the spinal cord cranially. The right T11 intercostal artery as well as the right L2 lumbar artery injection demonstrated a markedly hypertrophied right posterior spinal artery (PSA), supplying the ipsilateral portion of the AVM (figure 3B).

Endovascular treatment

A microcatheter was advanced into the largest sulco-commissure feeder from the anterior spinal artery (ASA) through the left T9 intercostal artery. Superselective digital subtraction angiography (DSA) demonstrated the AVM nidus with three venous aneurysms without opacification of the descending limb of the ASA. Provocative tests showed negative results; therefore this vessel was embolised using N-butyl-2-cyanoacrylate (NBCA) with occlusion of the venous aneurysms and preservation of the ASA distal to the malformation.

The right PSA was then superselectively catheterized through the right L2 lumbar artery. Superselective DSA revealed multiple feeding vessels from the PSA supplying the nidus. No normal PSA distal to the nidus was seen (figure 4A). At this point, a provocative test with 50mg of a short acting barbiturate (Brevitol) was negative. However, Xylocaine injection (40 mg) resulted in a significant (>50%) decrease of CSEP amplitude, starting one minute and with maximum decrement four minutes after injection and recovery to the baseline in approximately seven minutes (figure 1; Xylocaine test I). The test was repeated

with 20 mg of Xylocaine and again resulted in significant decrease of CSEP amplitude with a similar recovery pattern (figure 1; Xylocaine test II). No mMEPs changes were observed. The neuroradiologist performed a blind-test by injecting saline through the same catheter, while the neurophysiologist was not aware of the injected solution: no changes in CSEPs were recorded (figure 1; Saline test). The microcatheter was then brought back proximally within the axis of the posterior spinal artery and 40 mg Xylocaine was injected through this catheter positioning. Once more Xylocaine injection was followed by significant decrease in CSEP amplitude (figure 1; Xylocaine test III) without changes in mMEPs.

The repeatedly positive Xylocaine tests warranted further investigation of the vascular anatomy of the AVM and the lower spinal cord.

The microcatheter was further advanced in the PSA to the level of the distal portion of the nidus. Superselective DSA examination revealed the normal right PSA distal to the nidus as well as the left PSA through the anastomotic vessels (figure 4B). Existence of these normal vessels was considered to be the cause for the positive Xylocaine test. The distal normal PSA was protected by placing a coil and the microcatheter was then slightly brought back (figure 4C). Superselective DSA examination demonstrated the AVM without opacification of the normal PSA (figure 4D). At this point, provocative test was repeated by injection of 20mg of Xylocaine, but no changes in CSEPs were observed (figure 1; Xylocaine test IV); mMEPs remained unchanged. The vessel was then embolised using NBCA without subsequent changes in CSEPs (figure 1; embolisation). The control angiogram showed that opacification of the AVM, supplied by feeders coming off proximal to the embolised segment of the PSA, was significantly decreased but still present. Normal PSAs distal to the nidus were seen preserved (figure 4E).

No changes in CSEPs, mMEPs or BCR were observed at the end of the procedure. After the procedure, the patient inconsistently complained of slight increase in numbness in the lateral aspects of the right leg for several days.

Five days later, the second stage embolisation was performed. The same neurophysiological monitoring was used. Opening baselines from CSEPs, mMEPs and BCR, were very similar to the closing baselines of the previous procedure. In particular, with regard to mMEPs, left and right ABH and left TA mMEPs were present, while right TA mMEP was not.

The ASA was superselectively catheterized to the origin of the sulcocommisure artery feeder through the left T9 intercostal artery. Superselective angiogram demonstrated the distal ASA to the basket opacifying the bilateral distal PSAs as well as the remaining nidus of the AVM. Multiple attempts to catheterize this sulco-commissure feeder failed because of the sharp angle at its origin. Superselective DSA demonstrated reversal of flow in the ASA distal to the origin of this sulco-commissure feeder (figure 5A). At this point provocative tests were negative. The vessel was then embolised using a small amount of NBCA, which penetrated into the nidus. Control angiogram of the left T9 intercostal artery showed more stagnant flow within the nidus with preservation of the ASA axis (figure 5B). Angiogram of the right T11 intercostal artery showed further decreased opacification of the nidus (figure 5C).

The ASA was then infused with heparinized saline because of mild spastic changes seen on the control angiogram. A few minutes after embolisation, appearance of mMEPs from the right TA was noted (figure 6). This response became persistent by the end of the procedure (figure 7). The patient woke up from anesthesia without new neurological deficits. Despite the improvement in mMEPs from the right leg, no significant clinical changes in the distal right extremity strength were noted in the first month after embolisation.

Discussion

This case illustrates the usefulness of a multimodality neurophysiological monitoring when dealing with complex derangements of spinal cord vascularization such as intramedullary AVMs.

During endovascular procedures for the treatment of spinal cord AVMs, a detailed anatomical analysis of the angioarchitecture of the AVM and of the cord blood supply is mandatory. In association, however, neurophysiological monitoring offers a unique opportunity to investigate normal and pathological haemodynamic patterns in the spinal cord.

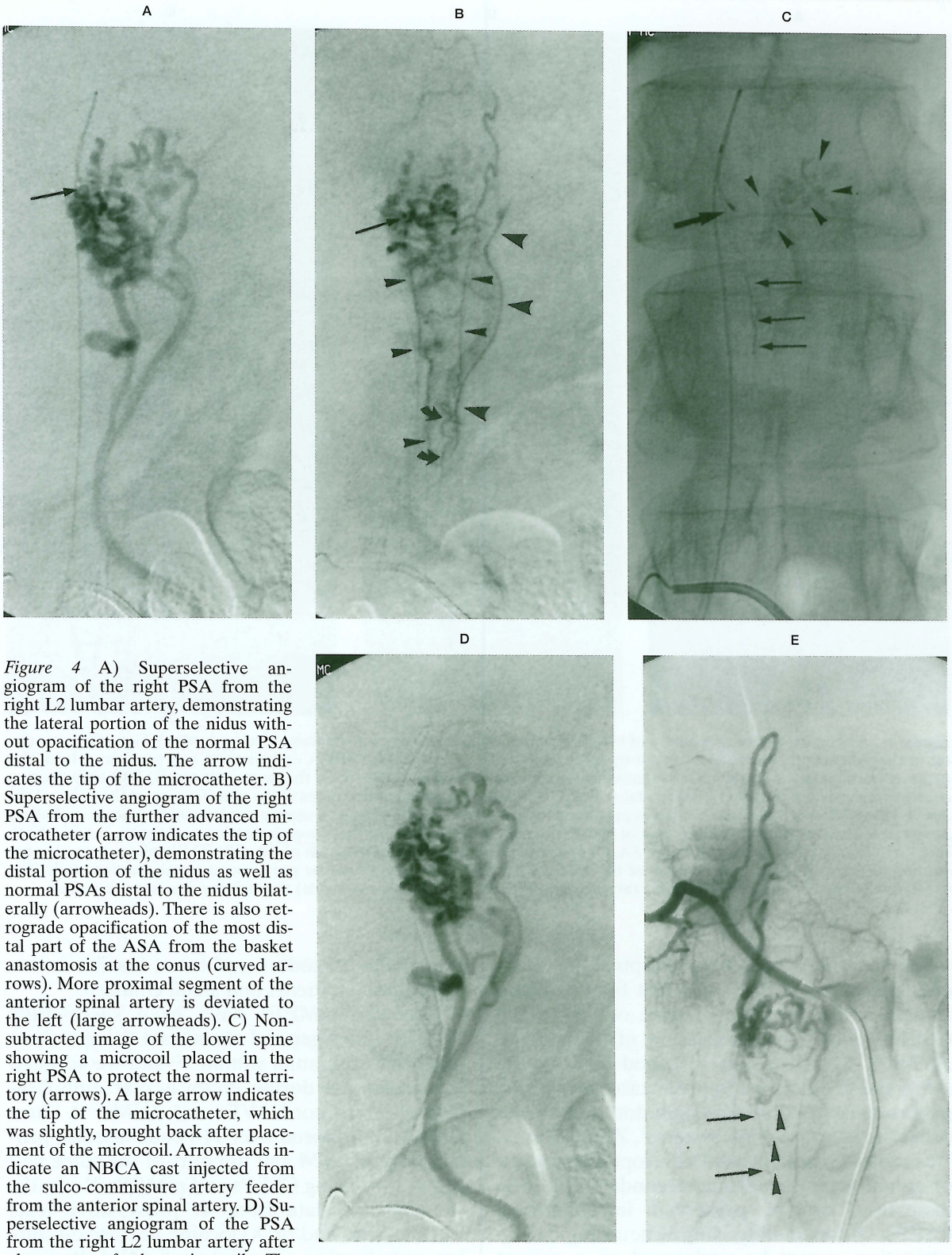

Figure 4 A) Superselective angiogram of the right PSA from the right L2 lumbar artery, demonstrating the lateral portion of the nidus without opacification of the normal PSA distal to the nidus. The arrow indicates the tip of the microcatheter. B) Superselective angiogram of the right PSA from the further advanced microcatheter (arrow indicates the tip of the microcatheter), demonstrating the distal portion of the nidus as well as normal PSAs distal to the nidus bilaterally (arrowheads). There is also retrograde opacification of the most distal part of the ASA from the basket anastomosis at the conus (curved arrows). More proximal segment of the anterior spinal artery is deviated to the left (large arrowheads). C) Non-subtracted image of the lower spine showing a microcoil placed in the right PSA to protect the normal territory (arrows). A large arrow indicates the tip of the microcatheter, which was slightly, brought back after placement of the microcoil. Arrowheads indicate an NBCA cast injected from the sulco-commissure artery feeder from the anterior spinal artery. D) Superselective angiogram of the PSA from the right L2 lumbar artery after placement of the microcoil. The catheter tip is at the same position as that in the figure 4C Embolisation was performed from this catheter position based on the negative provocative test. E) Post-embolisation control angiogram of the right T11 lumbar artery demonstrating decreased but persistent opacification of the nidus of the malformation, mainly proximal to the tip of the microcatheter. The right PSA distal to the nidus (arrows) is opacified through the anastomotic vessels (arrowheads) from the left PSA.

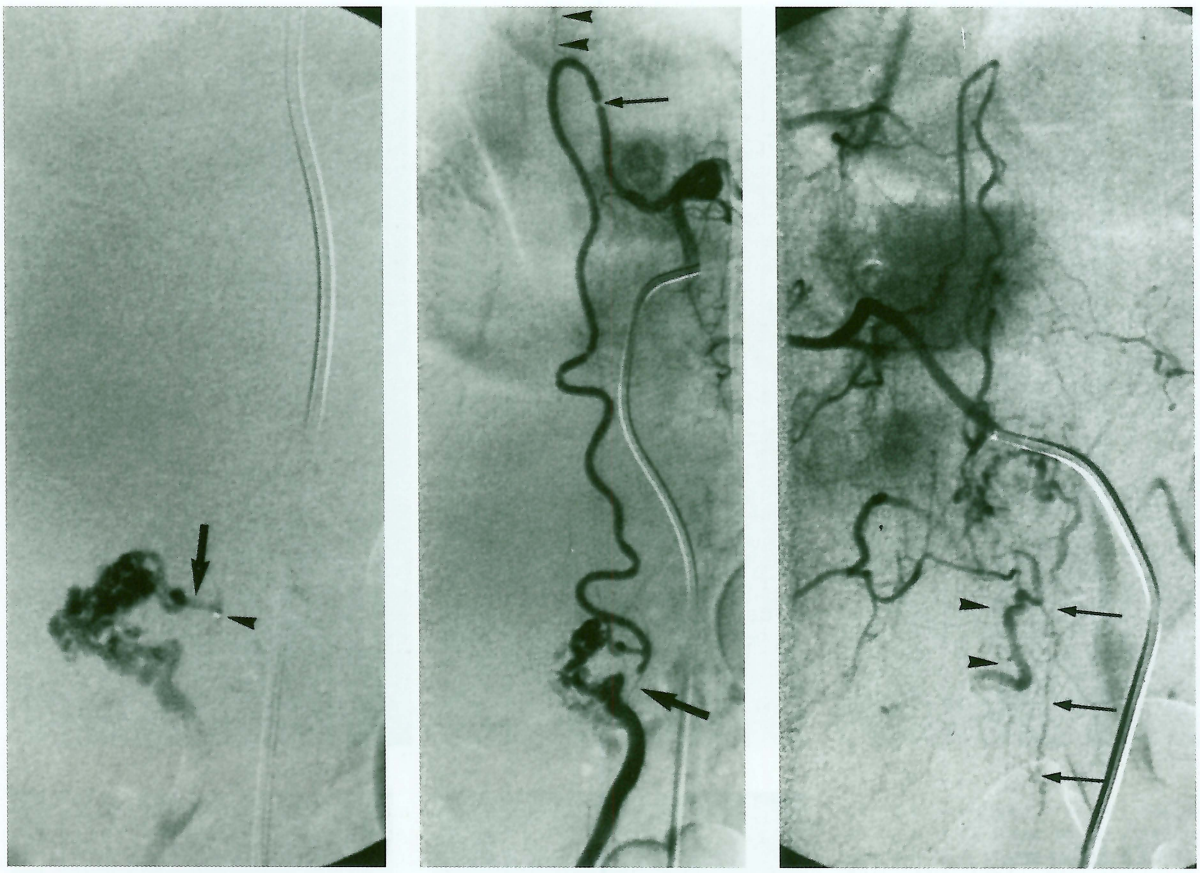

Figure 5 A) Superselective angiogram of the ASA at the origin of the sulco-commissure feeder (arrow). The spinal axis distal to the sulco-commissure feeder is not opacified due to the reversal flow. NBCA embolisation was performed from this catheter position based on the negative provocative test. Arrowhead indicates tip of the microcatheter. B) Post-embolisation control angiogram of the left T9 intercostal artery demonstrating significant decreases in the opacification of the nidus of the AVM. The ASA axis distal to the nidus is preserved (arrow). Mild spastic change is seen in the proximal radiculo-medullary artery (small arrow). The ascending limb of the ASA, which was not seen on the pre-embolisation angiogram, is now visualized (arrowheads). Compare with figure 3A. C) Post-embolisation control angiogram of the right T11 lumbar artery, demonstrating further decreased opacification of the nidus of the AVM compared with the previous post-embolisation study (figure 4E). The left PSA distal to the nidus (arrows) opacify the right PSA (arrowheads) through the anastomotic vessels.

Complexity and variability of spinal cord vascularization most likely account for deterioration in motor function in spite of unchanged CSEPs following embolisation of spinal cord AVMs [10]. The direction of blood flow in the ASA and PSA can be unpredictable and blood perfusion of the dorsal column does not always reflect that in the ASA system [17]. Zornow et Al [18] described a patient who developed an anterior spinal artery syndrome secondary to aortic dissection, despite preserved intraoperative CSEPs. Unfortunately, clinical neurological examination cannot be performed on a patient under general anesthesia unless the patient is wakened from anesthesia. However, this so called wake-up test causes significant prolongation of the procedure and is not feasible in uncooperative patients and small children.

Recently, mMEPs have been successfully elicited under general anesthesia using a short train of transcranial stimuli [19,20] and we adopted this technique during endovascular embolisation of spinal cord AVFs and AVMs [13,14]. Since 1996 we monitored over 110 endovascular procedures for AVM and tumours of the spine and spinal cord using CSEPs and mMEPs. Recently, we analyzed data from 49 provocative tests with Amytal and/or Xylocaine performed during embolisation of spinal cord AVM. Preliminary results suggest that mMEPs are more frequently affected than CSEPs by provocative tests when Xylocaine is injected either in the

ASA or the PSA; it is noteworthy that in the majority of cases disappearance of mMEPs was not associated with CSEPs changes [21]. Therefore, embolisation relying on provocative tests using only CSEPs would have resulted in postoperative motor deficits in some cases.

Unpredictability of provocative tests when Xylocaine is injected in the ASA and/or PSA supports the existence of vascular anastomoses and the variability of the spinal cord flow dynamic, this latter being even more complex in the presence of an AVM. The ASA territory can be infused by an injected drug through the AVM or through the hyperemic anastomotic vessels in the normal spinal cord around the nidus of the AVM. The usual PSA territory may not be infused depending on the position of the tip of the microcatheter and flow dynamics within the feeder to the malformation which has a preferential flow due to lower pressure. The watershed area between the ASA and the PSA can also be shifted due to the existence of an AVM.

In the presented case, we monitored CSEPs and mMEPs during a procedure involving both ASA and PSA territories. Based on the knowledge of vascular anatomy and neurophysiology [7,17], it is obvious that having mMEP monitoring is preferable for endovascular procedures through the ASA. This has been confirmed by Touho et Al [22] who described motor, but not sensory deficits as a result of Xylocaine injection in the ASA. Accordingly, in the first procedure we described, persistence of mMEPs after provocative tests encouraged embolisation of the anterior sulco-commissure feeder to the AVM. Although monitoring CSEPs for embolisation through the ASA may sound superfluous, we have experienced one case, out of 60 patients studied after provocative tests, where injection of Amytal/Xylocaine in the ASA caused loss of CSEP while mMEPs remained unchanged. We therefore routinely monitor CSEPs as well as mMEPs even if the endovascular procedure is limited to the ASA territory.

It might be claimed that disappearance of CSEPs, in a patient whose sensory functions are preserved, is per se a contraindication to embolisation of that vessel, and there is no need for motor evoked responses. However, there are situations where CSEPs are unmonitorable due to the underlying pathology. Under these circumstances monitoring mMEPs during

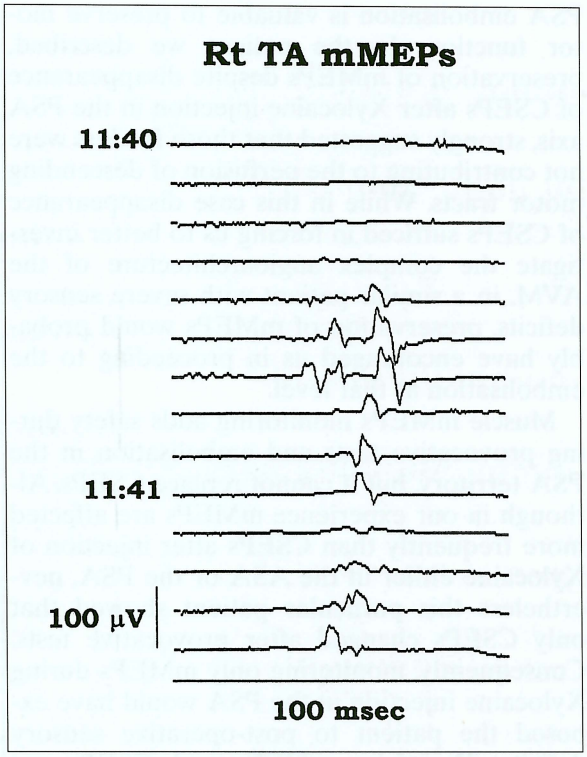

Figure 6 Appearance of the muscle MEP response from the right (Rt) TA muscle a few minutes after embolisation of the ASA feeder at T9.

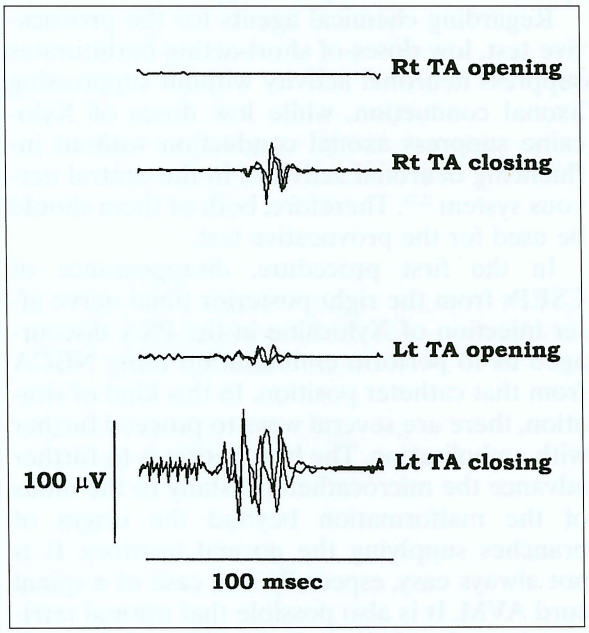

Figure 7 Muscle MEP from left (Lt) TA muscle was present at the beginning (opening) and at the end (closing) of the second endovascular procedure. The response from the right (Rt) TA muscle was absent at the beginning (opening) but appeared after embolisation and persisted until the end of the procedure (closing) (compare with figure 2B).

PSA embolisation is valuable to preserve motor function. In the patient we described, preservation of mMEPs despite disappearance of CSEPs after Xylocaine injection in the PSA axis, strongly suggested that those feeders were not contributing to the perfusion of descending motor tracts. While in this case disappearance of CSEPs sufficed in forcing us to better investigate the complex angioarchitecture of the AVM, in a similar patient with severe sensory deficits, preservation of mMEPs would probably have encouraged us in proceeding to the embolisation at that level.

Muscle mMEPs monitoring adds safety during provocative tests and embolisation in the PSA territory, but it cannot replace CSEPs. Although in our experience mMEPs are affected more frequently than CSEPs after injection of Xylocaine either in the ASA or the PSA, nevertheless this particular patient showed that only CSEPs changed after provocative tests. Consequently, monitoring only mMEPs during Xylocaine injection in the PSA would have exposed the patient to post-operative sensory deficits. Therefore, mMEPs and CSEPs are complimentary to each other and both should be monitored all the time for embolisation of spinal cord vascular pathology.

Regarding chemical agents for the provocative test, low doses of short-acting barbiturates suppress neuronal activity without suppressing axonal conduction, while low doses of Xylocaine suppress axonal conduction without influencing neuronal activities in the central nervous system [23,24]. Therefore, both of them should be used for the provocative test.

In the first procedure, disappearance of CSEPs from the right posterior tibial nerve after injection of Xylocaine in the PSA discouraged us to perform embolisation using NBCA from that catheter position. In this kind of situation, there are several ways to proceed further with embolisation. The best option is to further advance the microcatheter distally to the nidus of the malformation beyond the origin of branches supplying the normal territory. It is not always easy, especially in a case of a spinal cord AVM. It is also possible that normal territory is perfused through the nidus of the malformation. The second option is to block the origin of the vessel supplying the normal territory using a coil and then embolise with NBCA. In this case, the territory distal to the coil should be supplied by collateral vessels, which is almost always the case for the PSA due to the rich pial network vessels on the surface of the spinal cord. This is the strategy we adopted for this case. By further advancing the microcatheter in the posterior spinal axis, we were able to demonstrate the existence of the PSA supply to the normal territory distal to the nidus of the malformation. Repeat provocative test after coil placement was also useful to confirm that the normal territory was no longer perfused from the catheterized vessel. If it is impossible to demonstrate normal territory supply by advancing the microcatheter, the third option is to decrease the concentration of NBCA or change the embolic material to particles to decrease penetration of the embolic material into the nidus as well as to the normal territory. This is a less effective but safer way of embolisation in this kind of situation. The final option is to abort the idea of embolising this vessel and to stop the procedure completely or to catheterize a different feeder. In order to select best possible option among the above, reliability of provocative test is essential. In this sense, the current case is paradigmatic to demonstrate reproducibility and sensitivity of the provocative test with CSEP and mMEP monitoring, which makes us comfortable to perform endovascular embolisation of the spinal cord AVMs under general anesthesia.

Because any interventional procedure may acutely modify the local haemodynamics, it is critical to repeat provocative tests for both CSEPs and MEPs after any maneuver. In this patient, after the normal PSA distal to the nidus was excluded through the coiling procedure, we repeated the Xylocaine test and, this time, CSEP did not change and mMEPs remained stable. Monitoring only CSEP would expose to the risk of motor deficits because the possibility of local haemodynamic changes cannot be ruled out. In another hypothetical patient, with a different local haemodynamic pattern, a similar procedure might have affected vascular supply to the descending motor tracts; therefore, mMEPs might have disappeared after provocative tests.

Another interesting topic is the value of CSEPs and mMEPs to predict the recovery of neurological function after embolisation. We previously described the prognostic role of mMEPs after embolisation of a spinal dural ar-

teriovenous fistula [14]. The appearance of mMEPs at the end of the procedure was followed by immediate subjective feeling of increased motor strength, which was followed by objective improvement. On the contrary, in this report, the appearance of mMEPs from the right TA at the end of the second procedure was not supported by clinical evidence of significant motor improvement at the four-week follow-up. However, he may still improve in his motor outcome, because clinical experience with embolisation of intramedullary AVM suggests that time required for recovery is variable. The observation of mMEPs reappearance after embolisation of spinal AVM is still anecdotal [14] and the prognostic role of mMEPs during these procedures should be supported by a large series and a longer follow-up. Nevertheless, while this case suggests that appearance of mMEPs may not correlate with clinical outcome in a short-term perspective, the prognostic role of mMEPs during spinal cord surgery has been well documented [14,15,25] and we might expect a similar correlation for spinal AVMs.

Conclusions

To favor either CSEPs or MEPs during endovascular procedures in the spinal cord is not justified by a solid scientific background. To rely only on one of these two modalities can be misleading and ultimately result in new postoperative neurological deficits. This case report illustrates that monitoring both CSEPs and mMEPs, combined with provocative tests, represents the most effective neurophysiological monitoring during embolisation of a spinal cord AVM under general anesthesia.

This method of monitoring and provocative testing cannot replace a careful analysis of the vascular anatomy of the normal spinal cord and the AVM. However, we believe that the combination of anatomical and neurophysiological data provides the safest embolisation of spinal cord AVMs.

Acknowledgments

We thank Marco Campos for his editorial assistance.

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